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EDITORIAL.

LET'S HEAR FROM YOU

Nothing encourages an editor more than comment on or criticism of his product. We would welcome criticism of this Journal past and present, and either constructive or destructive. There are some unfortunate individuals, who are so completely satisfied with their own work that they consider it perfect and keenly resent criticism. We flatter ourselves that we do not belong to that class. On the contrary we would be glad to publish critical correspondence, and to consider and adopt ideas which appear reasonable to us and our advisers. Therefore we would urge that you make your suggestions and conscientious objections known and take an active interest in the welfare of your Journal.

MEDICAL LIBRARIES TO WHICH UNDERGRADUATES IN TORONTO HAVE ACCESS.

(3) LIBRARY OF THE DEPARTMENT OF PATHOLOGY.

2nd Floor Pathology Building.

This library is a very useful reading room for students of pathology and bacteriology as well as those engaged in scientific medical research. It is less valuable for the study of clinical medicine and surgery. The current numbers and bound volumes of all the leading journals of pathology and bateriology are in this library. The J.A.M.A. and the B.M.J. and Lancet are in this library also. The leading French and German journals of pathology and bateriology of the university library are on the shelves. There is a good collection of standard text books which may be used in the library. Books may not be removed.

MISTURA

If you have read anything, even remotely, connected with medicine, which you think others might enjoy reading, Mistura will always be here to let the other readers of this journal know.

The Eye of Allah, from "Debits and Credits," Rudyard Kipling, (Prof. J. P. McM.)

A monk of St. Ilods' Abbey, in England, sometime before the Renaissance obtains in the course of his travels a primitive kind of microscope. The Abbot, himself a physician, and Roger of Salerno examine a drop of water from a stagnant pool and discuss the possible relation of the organisms seen to the causation of disease.

The Abbot finally smashes the microscope so that these "devils" may not be revealed to a world not ready to understand, them.

In the South Seas, Robert L. Stevenson, —W.S.K.)

The author makes some interesting observations on the ravages of disease among the Polynesians, and the factors involved in the rapid depopulation of certain islands.

"Homines ad Deos nulla re proprius accedunt quam salutem hominibus dando."— "In nothing do men so much resemble gods as in giving help to their fellow creatures."

—Cicero.

Address by the President of the Academy of Medicine to the Graduating Year*

THE Academy welcomes tonight as its guests the Students in Medicine at the University with the hope that there may be in this meeting, pleasure and profit for both the fellowship and their guests. Like a new draft to an old worn regiment, you come, soon to join in the world-old conflict with the forces of disease, with your hopes high and your equipment new and shining and, very probably, with a feeling of kindly tolerance for some of us who are old-fashioned enough to bear ancient and rusty weapons and to distrust a little the very latest high pressure modes of attack.

We, the Committee, have thought that you might be interested to-night in hearing something of post-graduate study, of the advantages to be derived, and of the most useful way of spending time which you may be fortunate enough to occupy in this way, so we have asked a number of doctors who have recently returned from abroad to recount their experiences and to offer their advice which we are sure will be of interest and value both to you and to the Fellowship at large.

Many of you students have been guests of the Academy's library and I take this opportunity of assuring you that it gives us the greatest pleasure to have you come here and make use of the facilities afforded and the assistance of our capable and obliging staff. To learn how to use a library, to familiarize yourselves with the original sources from which your teaching has been drawn and to develop the critical faculty as you consider the sometimes varying conclusions advanced by workers throughout the world of science, is a valuable asset. And in the future when such facilities are not at hand, you will know how to keep in touch with advance and how to build up your own libraries. We would like you too, to see the advantages to be gained from such association of men in the same line of work so that in the future you will join the Academy if here, or if not, the nearest local medical association and in that community of interest you will learn to know and value your colleagues, to draw help and inspiration from them and to share in their problems. For you owe a debt to those who have preceded you in the patient study of disease, a debt which you can repay only by passing on what you have learned or may learn.

In conclusion, let me remind you of Osler's Master Word "Work," not for the next four months—even the most popular young man or young woman will have little chance to lose sight of it now—but for the future, when you must add to your stock or you will find yourself knowing less in five years than you know now. Let me urge you too to bear in mind the dignity of your chosen profession, a profession in which man intrusts you with the lives of women and little children and where only your knowledge and skill stands between them and the Grim Reaper. How are we to answer our conscience if by culpable ignorance or neglect we have failed to do all that any man can do. And if when you go out from the pleasant safety of laboratory and hospital where skill and knowledge can summon to your aid the most perfect scientific equipment, you are cast on your own resources—now seeming so pitiably inadequate, you can only go bravely forward knowing that the personal equation is the most important factor in the problems of medicine and think of what Sir James Mackenzie accomplished at Burnly, and take as your motto Browning's words:

"For more is not reserved
To man, with soul just nerved
To act tomorrow what he learned today.
Here, work enough to watch
The Master work and catch
Hints of the proper craft-tricks of the
tool's true play."

—A. J. Mackenzie.

*Read before the Academy of Medicine, Toronto, on Students' Night, January 27th, 1927.

The Parathyroid Glands

J. B. COLLIP
Professor of Biochemistry, University of Alberta.

THE parathyroid glands in the human subject are minute structures 3 to 15 mm. long and 2 to 3 mm. broad and thick. There are, as a general rule four distinct glandules situated in such relation to the thyroid lobes that it is customary to distinguish between a superior and inferior pair. Accessory glandules are sometimes present, and these may be situated in the thorax near to or imbedded in the thymus gland.

It was not until as late as 1880 that the parathyroid glands were discovered. Sandstrom at this time described the superior pair while Kohn in 1895 discovered the inferior pair. The parathyroid glands have since been found in all vertebrates with the exception of fishes.

Of late years the physiology of the parathyroid glands has been greatly elucidated as a result of numerous researches by many workers. They are primarily organs of internal secretion and they play a very important role in the regulation of the metabolism of calcium.

Removal of the glands results, as a rule, in death in from two to seven days. The symptoms manifested by the parathyroidectomized animal are well described by Vassale and Generali, who in 1900 removed the parathyroids from 10 cats and 9 dogs. Referring to the cats they state: "All of these presented a typical syndrome, viz., fibrillary contractions and muscular spasms, psychical depression, rigid and uncertain gait, anorexia, tachycardia, rapid emaciation, fall in body temperature and death," whereas they describe the results with the parathyroidectomized dogs as follows: "All were in good condition on the day following the operation. Symptoms began to appear on the second or third day and death resulted shortly thereafter. The symptoms were tremors, psychical depression, paresis of the muscles of mastication, trismus, rigidity of the hind limbs, uncertain and spastic gait, muscular weakness and convulsions. There was loss of appetite and vomiting, palpitation and dyspnoea."

W. G. MacCallum in 1909 showed that the blood calcium content became reduced following parathyroidectomy, and when it had fallen to approximately 50 per cent. of the normal value, that tetany appeared. He also demonstrated that the administration of calcium salts would give temporary relief from the tetanic attacks.

There have been two main theories of parathyroid function, one the detoxication theory, the other the calcium theory. According to the former hypothesis death from parathyroidectomy is due to poisoning by some product of metabolism normally detoxicated by the parathyroid glands. Guanidine compounds have been held by the Glasgow school to be the specific toxic reagents.

Guanidine salts injected into normal animals produce a type of fatal tetany which is in some respects similar to true parathyroid tetany. It is also claimed that there is also an increase of guanidine in the blood and urine in parathyroidectomized animals. The value of these results has recently been called into question. The low blood calcium which is a constant finding in parathyroid tetany is looked upon as a secondary phenomenon by the adherents of this theory, whereas it is considered the primary cause of tetany by those who hold to the calcium theory.

Various other factors which are of direct significance in this problem have been urged from time to time as of great or little importance in the pathogenesis of tetany. Chief among these are alimentary factors and disturbed acid base balance.

Alimentary factors of possible significance are:

(1) Diet.
(2) Type of intestinal flora.
(3) Absorption of excretion.

1. **Diet.**—It is an old observation that a meat diet is provocative of tetany, and a bread and milk diet of definite value in prolonging the life of parathyroidectomized ani-

mals. Greenwald has pointed out that the precipitation of tetany by meat feeding is in reality due to the phosphate in the meat. The blood calcium falls in parathyroidectomized animals and the inorganic phosphorus in the blood rises slightly as a rule. Any sudden increase in the inorganic phosphorus of the blood will precipitate a violent attack of tetany.

2. **Intestinal Flora.**—The reaction of the intestinal contents is influenced by the type of flora present. An aciduric flora is beneficial either by way of increasing the absorption of calcium or in reducing the production and absorption of exogenous toxins. A milk diet with lactose and dextrin added tends to produce an aciduric flora in the intestine and its usefulness is probably dependent upon this result.

3. **Absorption and Excretion.**—Calcium is normally absorbed from the upper intestinal tract and excreted into the lower part. Any method which will keep the blood calcium within or near to normal limits will control or prevent tetany in parathyroidectomized animals. Among such methods which have been successfully used is that of the repeated injection or ingestion of calcium salts. Injected calcium rapidly leaves the circulation in both the normal and parathyroidectomized animal. During the time that the calcium content of the blood is elevated as a result of calcium therapy in the parathyroidectomized animal a normal condition is manifested. Calcium therapy is only palliative, as it does not in any way restore the power of the body to maintain a normal level of blood calcium. The time when this treatment can be safely abandoned is probably the time when accessory glands have developed sufficiently to produce enough hormone to meet the minimum requirements of the organism. The permeability of the mucosa of the bowel to calcium is an important factor both as regards calcium absorption and excretion. To what degree this may be influenced by both endogenous and exogenous factors is not yet determined.

Acid-base Balance.—Normal neuro-muscular tone is definitely related among other things to the normal inorganic blood picture. The inorganic elements and radicals are effective as ions rather than as atoms or molecules of any substance, for example, is directly related to the strength of the solution. It is also influenced by other factors such as temperature and reaction. In the case of calcium, ionization is greatly affected by changing the hydrogen ion concentration. Any factor which will cause a decrease in the pH of the blood will increase the ionization of calcium and any factor which will cause an increase in pH will cause a decrease in the ionization of calcium. Thus tetany may be associated with a condition of alkalosis and the blood serum calcium be normal. The ionized calcium must, however, be reduced in such cases. The tetany produced by overventilation of the lungs is of this type. Hyperpnoea is sometimes manifested after parathyroidectomy. Animals showing this symptom have the most severe type of tetany. Ammonium chloride has recently been successfully used in the treatment of parathyroid tetany. Here the action is probably that of an acid increasing the ionic calcium in the blood stream. In general it may be said that alkalis tend to precipitate tetany, and acids to counteract or prevent it.

The Parathyroid Hormone.—The parathyroid hormone, which has been prepared in stable form by extracting the fresh parathyroid glands of oxen with dilute boiling hydrochloric acid and purified by appropriate means, has the following physiological properties:

1. It will entirely prevent tetany in completely parathyroidectomized dogs treated by subcutaneous injections of adequate dosage.

2. It will control tetany of parathyroid orgin. Relief is afforded in a few minutes by intravenous injection of the hormone.

3. It raises the blood serum calcium in both normal and parathyroidectomized animals.

4. It can be biologically standardized by determining its calcium mobilizing power in normal dogs.

5. It will produce fatal hypercalcaemia in normal or parathyroidectomized dogs submitted to overdosage.

6. A unit is defined as 1/100th of the amount which will produce a 50 per cent. rise in the blood serum calcium of normal dogs weighing 20 Kilos.

Congenital Hypertrophic Pyloric Stenosis

L. A. MACKLIN, 2T7.

THE term hypertrophic suffices to suggest the pathological picture. The term congenital indicates the time of origin of the lesion—for it is generally considered that this lesion must have started considerably prior to birth.

This condition of the pylorus is a mechanical obstruction of the alimentary tract —due to a simple hypertrophy and hyperplasia of the circular muscle fibres of the pylorus.

History.

The credit for first considering the pathological picture in such cases is awarded to Hirschsprung whose works in 1888 seem to have initiated clinical reports of similar cases recognized during life.

Landerer in 1879, P. Maier in 1885 accurately described specimens of pylori from adults—who died from other diseases showing no relation to the gastric condition. The descriptions of the specimens accurately portray the findings of today. "The tumors were hard muscle masses with lumens 3-5 mm., and the pyloric antrum was funnel shaped." These patients had a history of always taking their food in measured amounts.

What is regarded as the first paper on the subject is that reported by Beardsley in 1788 under the title "Scirrhus of the Pylorus in an Infant." This paper was brought to light by Sir Wm. Osler.

Recent attention has been called to the text book by Geo. Armstrong—an English paediatrician of 1777—in which he accurately described a typical case and reported several others.

As stated above, the modern history of hypertrophic pyloric stenosis began following Hirschsprung's contribution in 1888, although not for several years did the subject appear strongly to impress the medical profession. In 1898—ten years later—Cautley was able to record twenty cases, including two of his own; and in 1902 recorded fifty more cases, in which nineteen had been operated on. During the last twenty years this condition has taken its place as an important disease in childhood ranking with appendicitis and intestinal obstruction in seriousness and in frequency.

Etiology .

The etiology of congenital hypertrophic pyloric stenosis is entirely in the realm of conjecture. It occurs much more frequently in males 3:1 or 4:1. A large percentage of the cases occur in the first-born and also in some instances the second and third child show a similar condition. Similar conditions have been noted in six and seven month fetuses.

That hypertrophy follows over activity on the part of the stomach—due either to altered secretions or abnormal nerve impulses must obviously be pure conjecture.

In studied cases the stomach secretions have been normal.

To hyperperistalsis found after the lesion has occurred can hardly be ascribed any significance other than that of a secondary role.

One difficulty in determining the etiology of the lesion is our inability to determine the time of its origin. On one hand the clinical symptoms are rarely present during the first days of life. This has been considered an indication of post-natal development. However, the tumor when found is of such size that it can scarcely be credited with forming during the short period of its clinical presence. Cautley's recognition of the condition in a six months fetus undoubtedly fixes the possibility of its congenital development. Experimental attempts with adrenalin to reproduce the picture in animals have failed.

Pathology.

The pathological picture presents a remarkable uniformity. The essential lesion is described as an hypertrophy and hyperplasia of the musculature of the pylorus and in particular of its circular fibres. The other changes are practically all secondary to the obstruction.

Macroscopically the pylorus appears as

a hard rounded olive-shaped tumor mass covered by normal glistening peritoneum and sharply demarcated from the distended stomach above and the empty duodenum beyond.

It has the firmness of cartilaginous tissue. It is remarkably white and bloodless. It is freely movable with no peritoneal adhesions.

On longitudinal sections, it presents a dense firm uniform structure down to the mucosa.

The mucosa when freed from the constriction of the tumor mass seems normal.

Inflammatory change has rarely been seen and is probably secondary to the retention of food and the traumatism resulting from hyperperistalsis.

The stomach is uniformly dilated and usually normal in appearance though in some instances it seems thickened.

The duodenum is flat in sharp contrast to the distended stomach above.

Bernheim is quoted as saying that the nuclei of the muscle cells are larger in all dimensions than those in normal pylori.

Symptoms.

The essential features of the lesion present striking uniformity. In most cases the onset has been from two to four weeks after birth. It is always abrupt—its onset usually determined to the day.

The first symptom has been vomiting accompanied from the start by constipation and followed very soon by a startling loss in weight. These symptoms are usually progressive, never letting up for more than a few hours at a time.

In none of the cases has there been anything to indicate the oncoming trouble.

(1.) Vomiting—The initial vomiting is not characteristic, usually starting as a "spitting up." However, once started, the vomiting is characteristically explosive or projectile in type and occurs following the taking of food—not during the feeding as is seen in oesophageal obstruction.

When a meal or two is passed without vomiting, the next vomiting is likely to result in the return of the accumulation of the several past feedings. The vomiting is not associated with any apparent nausea, the child often being eager for its food.

The vomitus is bile-free and often shows hyperacidity. It contains stomach contents and nothing more.

(2.) Constipation—Vomiting in young children is apt to be associated with diarrhoea, but the vomiting in pyloric stenosis, being caused by obstruction, is associated with marked constipation, which in advanced cases may be described as absolute.

The persistence of bile in the stool serves to eliminate the possibility of obstruction lower down.

(3.) The obstruction is so high that fluids also are not retained and this aids in causing a perverted body metabolism and acidosis sets in, with dehydration and amazingly rapid loss in weight—which may be as much at 35 to 40 per cent. in 2½ to 3 weeks time.

(4.) Urine—Becomes concentrated—highly acid and brick-red uric acid is present in great concentration.

(5.) Fever—There is no rise in temperature until later on in the condition—when secondary infection is apt to set in.

Physical Signs

The child rapidly assumes an emaciated appearance—though at first it looks healthy and comfortable.

The baby's abdomen presents characteristic features. The upper part of the abdomen is full, tense and bulging, and is made more prominent by a marked retraction of the hypograstric region.

Slowly moving peristaltic waves cross the upper abdomen from left to right—their size and frequency being increased by ingestion of food and water.

The abdominal walls are relaxed—there being no suggestion of rigidity. The stomach appears to be a bulging cystic tumor easily palpated.

In the cases of Scudder, Downes, etc., the pyloric tumor was present in 92 per cent. of cases easily palpated and thus is of great diagnostic importance.

The emptying time of the stomach is greatly delayed and in some cases there is absolute retention.

X-Ray Examination.

This is of value merely in confirming a provisional diagnosis.

The opaque meal should be given by

bottle or stomach tube, and the stomach fluoroscopically examined from time to time.

It will be noted that the pyloric end of the stomach rounds out, but maintains a sharp clear outline.

Stomach Contents.

The chemistry of the stomach varies within normal limits, with perhaps a tendency to hyperchlorhydria. The bacterial flora present nothing of note.

Differential Diagnosis.

(1.) Pyloric stenosis is diagnosed from intestinal atresias by the interval of some days ere the symptoms develop.

(2) The time factor and also the type of vomiting and the absence of tumor or dilated stomach eliminates atresia of the oesophagus.

(3.) Cardio spasm may be present from birth or develop later. Besides the absence of the abdominal signs, the vomiting is milder and occurs during the feeding process.

(4.) Congenital kinking of the duodenum presents a condition indistinguishable from pyloric stenosis, except the absence of a tumor mass.

(5.) Congenital retroperitoneal hernias have presented obstructive symptoms, but the mass is fixed, is larger, and is softer than a pyloric tumor.

(6.) Pyloric-spasm—This is taken up at the end of this discussion.

TREATMENT.

General Consideration.

The surgical treatment of pyloric stenosis in infants is based essentially on mechanical conditions underlying the clinical picture. The obstruction is sufficient to produce death from starvation long before any natural tendency to spontaneous resolution manifests itself. No similar obstruction at any other site in the gastro-intestinal tract either in adult or child would be treated by any other than surgical means In the opinion of Abt, the tumor, during the operation, has always presented so satisfactory a reason for obstruction that the element of spasm is a very minor factor. The use of atropine has been frequently found of great value—in permitting the emptying of the stomach—and the theory that spasm plays a role has been strengthened on the basis of the physiological action of the drug.

The obstruction is unassociated with circulatory changes as are seen in strangulated hernias, volvulus, intussusception, etc., and this explains the continuance of life in cases of obstructed pylorus.

The indication for operation is present when the diagnosis is made, and though several decades ago prelininary dietetic and medicinal treatment were considered essential, it has been proved that early operation increases the chance for life and minimizes post-operative convalescence.

Pre-Operative Preparation.

1, starvation acidosis; 2, water deprivation, 3, loss of heat during operation, and 4, 3, loss of heat during operation and 4, operative shock, constitute the immediate menace to the patient.

Since liquids do not pass the pylorus, the infant should receive fluid interstitially, or by rectum, or intravenously. Infants accept fluid by rectum very poorly.

The best fluid, in my mind, is normal saline to overcome dehydration and containing 10 per cent. glucose to overcome the acidosis. If given intravenously 500 cc. may be given. By interstitial only that amount required will be absorbed. The work of Mattill has shown that the glucose tolerance in such infants is very high. Mattill showed that:—

(1.) The glucose tolerance of the normal child as determined by the Woodyatt method is practically identical with that of the average adult which is 0.8 to 0.9 grams per kilo per hour.

(2.) The tolerance of atrophic infants for glucose is considerably greater—it varied in a series of thirty-two cases from 1.4 to 1.8 grams per kilo per hour.

Immediately preceding the operation the child should be well wrapped to prevent loss of heat. The operating room should be warm.

The stomach should be emptied of solids and gas to reduce distension.

Ethyl chloride induction followed by ether is found to be the safest mode of anaesthetizing the child.

Operative Methods.

There have been several methods of dealing with these cases, but I will only deal with one for I consider it better to know one than to think of several. As Wallstein said, after studying the healing process of the pylorus after the Fredet-Rammstedt operation, "healing is brought about by the cells of the serosa and submucosa, but the non-striped muscle cells have no part in the process. The wound in the pylorus is healed within nine days. The pylorus has become relaxed in two weeks. The stomach has returned to its normal size in one month. The gap between the cut surfaces healed in six weeks. In two years, only a line of fibrous connective tissue separates these two muscle ends, and the stomach is quite normal. The Fredet-Rammstedt operation cures the pyloric lesion."

This modified pyloroplasty as carried out by Rammstedt consists in making a longitudinal incision along the anterior surface of the tumor, permitting the retraction of the tumor mass and the freeing of the mucous membrane.

The incision should not be carried down to the mucosa for fear of cutting it, but a sharp nosed artery forcep introduced and the muscle mass spread. The muscle tends to split readily. The whole canal should be so exposed and all muscle fibres broken.

In some cases surgeons prefer to suture the right border of the omentum which is easily accessible to the lines of incision through the serosa.

No modification of the Rammstedt operation is worthy of description, since it is simple; relieving the obstruction in the simplest manner, requiring the smallest amount of time and manipulation, and with almost perfect post-operative results.

Post Operative Care.

The operation is followed by a blood transfusion of 15cc. per pound of body weight in every case.

The infant on its return to the ward is placed on its right side and the head of the bed elevated after recovery from anaesthetic. Four hours after operation one half ounce of full strength breast milk is given. The patient is then fed every four hours and the amount increased one dram at each feeding. Usually by the third day the infant can be nursed. Some vomiting may occur for three or four days after the operation. There are no surgical contra-indications to early feedings, although the condition of the child may constitute such.

Prognosis Mortality.

Of factors affecting mortality most surgeons agree that delay in operating is the greatest cause of death due to excessive loss in weight and tissue changes associated with starvation and dehydration.

Spence, from a series of 163 cases, concludes that:—

(1.) When symptoms have lasted less than four weeks, the mortality is only one third as great as when the symptoms are over four weeks.

(2.) In infants weighing 7 lbs. or less, the mortality was 3½ times as great as in those weighing over 7½ lbs.

(3.) The mortality increases in direct proportion to the amount of weight lost previous to operation.

Medical Treatment.

Three main methods have been employed in the medical treatment of fpyloric stenosis.

The first is the re-feeding of the infant after it has vomited. It has been observed by Findlay, Gerstley, Davison and others that infants who have vomited will frequently retain a second feeding if it is given immediately following the vomiting. Davison states, "The fact, that in many cases of pyloric stenosis, a palpable tumor can only be palpated immediately after a peristaltic wave, would seem to indicate that the hypertrophied pylorus is relaxed between gastric contractions." Because of this fact, feeding and re-feeding woman's milk is frequently successful. If food is administered persistently, eventually some of it may pass the hypertrophied pylorus during one of its periods of partial relaxation and in this way, the patient's nutrition may be maintained until the growth of the digestive tube compensates for the hypertrophy of the pylorus. There is no doubt that satisfactory results are obtained in a large percentage of cases by this method but the disadvantages are that a large amount of woman's milk is re-

quired which is frequently difficult to obtain. In addition a long stay in hospital is necessary, during which time the patient is in constant danger of acquiring some parenteral infection with disastrous results.

The second mode of medical treatment is in the administration of atropine sulphate just prior to each feeding. The most ardent advocate of this method is Haas who believes that pyloric stenosis is simply an advanced degree of pyloro spasm. He concludes that, "Most if not all cases can be cured by sufficient doses of atropine correctly administered." He starts with drop of a fresh 1 in 1000 solution of atropine with each feeding and increases the dose 1 drop each feeding or with each alternate feeding until vomiting ceases, or physiological effects are produced, occasionally having to give as much as 1/250 grain of atropine with each feeding. Beneficial effects have also been reported by Ibrahim, Johanssen and others. The Hospital for Sick Children results in treatment of pyloric stenosis with atropine having been disappointing, although in certain cases of pylorospasm or hypertonia the effect has been spectacular. Other observers elsewhere have not been impressed with the value of atropine.

The third method of medical treatment is the use of a thick cereal feeding advocated by Sauer. This feeding is prepared by cooking cooked cereal with milk, water, and if necessary sugar, until the mixture is of a thick consistency and barely drops off the inverted spoon. This feeding is then given to the infant by placing small portions on the end of a tongue depressor and scraping it off onto the back of the tongue with a second tongue depressor. Sauer found this method effective in twenty-eight of his thirty-five cases.

Porter reports ten cases treated in this manner with no mortality.

There is no doubt that the use of thick cereal feedings either alone or with atropine is an effective weapon in combatting vomiting due to pylorospasm, hypertonia and certain mild cases of pyloric stenosis. On the other hand, it was seen that a great number of cases of pyloris stenosis in which the use of thick cereal feedings in the interim between admission to hospital and operation gave disappointing results.

The series of patients recorded at the Hospital for Sick Children is the first large series from a Canadian source. It consists in 120 cases from 1920 to 1925. In every instance medical treatment was supplanted by surgery, the Fredet-Rammstedt operation being performed with a mortality rate for the whole series of 13.2 per cent. The diagnosis of pyloric stenosis was confirmed at operation in every case.

The general condition of seventy-five of these 120 cases was fairly good and the mortality in this group was only 8 per cent. In the other forty-five cases the condition was very poor and the mortality was 22.7 per cent.

Cause of Death.

The cause of death in the 16 cases was as follows: Decomposition, 5; respiratory infection,3; moribund on admission, 3; decomposition and diarrhea, 1; post operative acidosis, 1; general peritonitis, 1; volvulus, 1; collapse, 1.

Why Do We Submit All Our Cases of Pyloric Stenosis to Operation as Soon as a Definite Diagnisis is Made?

Sauer has collected from the literature a series of 497 cases who were given dietary treatment exclusively with a mortality rate of only 8.9 per cent. As mentioned before, there is little doubt that there are included with these medically treated cases many infants with pylorospasm, as some authors do not differentiate between the two conditions. It is the custom at the Hospital for Sick Children to classify infants with pylorospasm and hypertonia simply as difficult feeding cases and to treat them as such. Only one of these cases coming to autopsy during the past six years proved to be pyloric stenosis. Therefore, I believe that the vast majority of so-called medically treated cases of pyloris stenosis should only be classified as such when the tumor can be palpated. It is reasonable to conclude that the general mortality of the medically treated cases of pyloric stenosis osis is as high or much higher than the Hospital for Sick Children mortality rate of 13.7 per cent., in surgically treated cases, or rather taking the fairer average of the seventy-five patients in good condition of 8.2 per cent.

The hospital stay is much shorter with surgical than medical treatment. The average duration of hospital stay for surgical cases was sixteen days while that of medical cases was fifty-three days.

Although many infants who are in good condition may be medically treated with success, the long hospitalization with continued exposure to parenteral infections, the additional care, the long-continued anxiety of parents and physician, the large amount of breast milk required, all these combined with the low mortality rate of surgically treated cases in this group (8.2 per cent.) lead to the belief that it is advisable to submit all cases of congenital hypertrophic stenosis of the pylorus to operation.

Spasmodic Obstruction of the Pylorus.

No discussion of congenital hypertrophic stenosis of the pylorus is complete without considering a rather loosely defined group of cases described under the above caption, yet which are characterized by persistent vomiting.

The vomiting comes on later, usually in the second month. Instead of early becoming persistent and progressively more severe, it increases irregularly or abates for a while to return in an increased degree. This picture of variable vomiting may persist for five or six months. In the meantime sufficient nourishment is retained to allow the baby to hold its weight or even show a slight gain. The vomiting lacks the projectile character and is more of a spilling over, suggesting rumination.

The abdomen lacks the characteristic picture. The violent gastric peristaltic movements are usually absent.

There is no pyloric tumor present at any time.

The dehydration accompanying high intestinal obstruction is absent.

The stools are not typical starvation stools and the urine is fairly normal.

The presence of any true pathological condition at autopsy is absent. In a series of seven cases laparotomy revealed no lesion.

Treatment.

Here the treatment is essentially medical To perform a pyloroplasty on a normal pylorus would be utterly illogical.

The treatment most in vogue is thick cereal feeding combined with small doses of atropine sulphate.

The School of Salerno

EVELYN BRESLIN, 3T0.

THE history of the School of Salerno embraces a long chapter in history. Salernitan literature is comparatively abundant during the pre-scholastic age when the cultural history of the rest of Europe was very meagre, and since the history of this school is closely bound up with the Renaissance, anything that bears on its origin and literature must therefore be of value.

The Medical School at Salerno was the first European institution to develop an organization that can be called a University. It never developed any other branch of learning, however, and was nothing like modern universities. Three periods in its history can be distinguished—the Early Salernitan period, which corresponds to the period before its rise to fame; the Middle or High Salernitan period during which it was most famous; and then the Late Salernitan period or period of decline.

This School was never really founded. It was the natural product of certain very peculiar and complex surroundings. One legend says it was founded by four masters, a Greek, a Latin, an Arab and a Jew. This is not literally true, but corresponds to the mingled influences under which the School actually arose. As early as the tenth century Salerno was famous for its numerous physicians. The city of Salerno is an ancient seaport near Naples in the south of Italy. It was renowned as a health resort, and this and its mild climate may have been

the cause of its developing a School of Medicine through the gathering together of many sick patients, especially those of wealth.

From the eighth century to the fourteenth century there developed a peculiar type of Latin learning expressed in an old script called Beneventan, and the earliest medical documents known in this part of Southern Italy were in this script. These documents contain absolutely no original material. They are entirely translations and paraphrases or extracts of Greek; works slavishly copied and containing many errors. In these documents we have, therefore, static material passed on from hand to hand and degenerating in the passage.

The earliest Salernitan teacher of whom we have historical records, is called Gariopontus, who died about 1050 and is spoken of as a learned and skilful physician, but his work is just a patchwork of passages from old Latin translations of Galen and other ancient authors. In 1076 Salerno fell to the Normans, and under Norman influence it began to take on the appearance of a university. After the middle of the eleventh century the system of Medicine that was followed changed from methodism to the humorism based on Hippocrates and Galen and from this time dates the Medical Renaissance.

The zenith of the Medical reputation of Salerno is marked by the visit of Robert, Duke of Normandy, who came to the place to be cured of his wound after the Crusade of 1099. The celebrated medical treatise on Medicine or Hygienics called "Regimen Sanitatis Salerni" was dedicated to him as King of the English on this occasion. It passed through 240 editions and was translated into Irish, Bohemian, Provencial and Hebrew. Part of the English version is as follows:

"Salerno's school in conclave high unites
To counsel England's King, and thus indites:
If thou to health and vigor wouldst attain,
Shun mighty cares, all anger deem profane;
From heavy suppers and much wine abstain;
Nor trivial count it after pompous fare
To rise from table and to take the air.
Shun idle noonday slumbers, nor delay
The urgent calls of nature to obey.
These rules if thou wilt follow to the end,
Thy life to greater length thou mayst extend."

For more than two centuries Salerno as a School of Medicine stood fully on a level in point of academic fame with Bologna as a School of Law and Paris as the headquarters of scholasticism.

In 1231 Frederick II. as King of Sicily, forbade either the practice or teaching of Medicine within his dominions without the Royal License which was to be given after examination in the King's court by the Masters of Salerno and certain other Royal officers. The ceremony of graduation or promotion was performed in public and demanded of the candidate the fulfilment of the following preliminary conditions: seven years' study, the attainment of the age of twenty-one years, legitimate birth, a satisfactory examination in the Hippocratic, Galenic and Arabian writings, a promise to teach correctly and in accordance with the received doctrines, to administer no poisons, and to treat the poor gratuitously. These requirements fulfilled, the candidate received a ring, a wreath of laurel, and finally the benediction and thereafter he could teach and practice wherever he wished. The office of teacher was open to all graduates of this School as well as those of the later Universities.

The tendency of the School of Salerno was entirely and eminently practical, hence it laid great stress on symptomatology, dietetics, treatment and materia medica and less in accordance with the age, on physiology and human anatomy. Considerable importance was laid upon the manner of the physician, and some of the laws governing the physician's behaviour are to be found in the book entitled "The Coming of a Physician to His Patient." Some of the most important of these are: When the physician goes to visit his patient he should place himself under the protection of God and the Angel who accompanied Tobias. On his way he will try and learn from the person who came to fetch him as much as possible of the condition of the patient, so that if, after having examined the urine, and felt the pulse, he cannot soon learn the nature of the illness he can, by means of the facts

previously ascertained, at least inspire confidence in the patient by proving to him that he has divined something of the nature of his sufferings. It is well that the sick man should confess himself, or at least undertake to do so, because if his doctor finds it necessary for him he will believe his case desperate and the inquietude will aggravate his illness, whereas more than one sick man who provides against the reproaches of his conscience recovers because of his reconciliation with the Great Physician. On departing the physician promises the patient he shall recover; to those who are about the sick-bed, however, he must affirm that the patient is very ill; if the patient recovers the physician's reputation will be enhanced, should he die the physician can state that the outcome was as he had predicted.

Surgery was developed at Salerno to a very high level, and two of the most outstanding figures were, Roger of Palermo, who was considered the "Father of modern Surgery," and Roland of Parma, his disciple.

The anatomy studied was based on that of swine. Their physiology was Galenic. They believed in the four humours of the body—black bile, blood, phlegm and yellow bile. They described the four temperaments thus: sanguine people are jovial, the servants of Venus and favourites of Bacchus. Bilious people are cunning, their faces pitted and yellow; phlegmatics are described as being without desire and cherishing sleep. Melancholics are jealous to excess and laugh at the reverses of others, and are irritated by their successes. They believed that the heart has two cavities, the right receives the vena cava, and by large branches to each lung nourishes it. From the left side of the heart two arteries are given off, one to the lung, while the other carries the vital spirit with the blood illuminating the whole world of the human frame with vital rays. How remote from fact is the account. Thirty-two years later Harvey explained it and we can well agree with Bichat that "nothing is so plain as the discovery of yesterday, nothing so difficult as the discovery of to-morrow."

The downfall of the School was caused mainly by the growing influence of Arab culture which was introduced by Constantine the African, about whom many legends are told. The growth of other Universities such as Montpellier, also led to its downfall, and it was officially closed by Napoleon in 1817.

The profound importance of Salerno as regards Medical culture depends not upon any memorable contributions to science, but rather upon the fact that here the principles of the great physicians of antiquity were first cultivated independently and subsequently when received through the medium of the Arabians (though in a corrupted form) were carefully preserved. The School promised a free field of action for study since as far as was possible at that day it held itself aloof from the fetters of the fanatical church. Thus is attained an international importance, for here Arabians and Jews as well as Christians studied and interchanged ideas. It was like Alexandria, one of the historical bridges over which ancient culture and particularly ancient Medical culture, took its way during the Middle Ages from East to West. Salerno was also the medium by which Arabian pharmacy and therapeutics were introduced into the Medicine of the West. It is probable that it borrowed from the Arabians the principle of arrangement to transmit it again in the sequel to the European Universities. It also anticipated modern co-educational ideas by admitting women to its school both as teachers and students. There are many legends about the "Women of Salerno," the chief of whom was Trotula, whose name has descended to us and is now remembered as "Dame Trot."

The Salerno of to-day contains not a single book or manuscript of its ancient libraries, not a stone of its hospitals to commemorate its glorious past. The academy is not a ruin, nor even a memory, but exists only as a record of achievement, an ancient tale of departed greatness. The lines of Longfellow to Amalfi may well be applied here:

"Vanished like a fleet of cloud,
Like a passing trumpet blast
Are those splendours of the past."

IRITIS.

C. E. HILL, M.B.

IRITIS and Irido-cyclitis (Uvitis) should be considered conjointly. The iris and ciliary body form one continuous tissue because the iris is a prolongation forward of the ciliary body. Structurally, they both contain muscle fibres and are prone to any inflammation peculiar to such tissue. They also contain glands, and as stated previously, are prone to inflammation that any gland may have. They form the aqueous and by their endothelial surface can take up phagocytic action when infection is present.

The symptoms in general are hyperaemia of the iris which is shown by discoloration and loss in lustre, contracture of the pupil, which becomes irregular and bound to the lens by adhesions (Synechiae), circumcorneal injection, sensitiveness to light (photophobia), lacrimation, and exudation into the anterior chamber which can either form a false pupillary membrane or a corneal opacity due to deposits, which are "plastered" on the posterior corneal surface in a pyramidal shape with heavier deposits at the base and finer ones at the apex.

The subjective symptoms are loss of sight due to exudates with, as we have stated before, lacrimation and pain. There are symptoms peculiar to the etiological cause, namely:

Syphilitic Iritis gives at onset little or no pain, but there is gradual loss of sight and a minimum amount of ciliary injection (circumcorneal).

Diabetic Iritis has a more rapid onset, usually more exudates than in lues, considerably more ciliary injection, and more liability to secondary glaucoma than in the luetic type.

The so-called Rheumatic type is due to a focus of infection such as teeth, tonsils, or sinuses. If they have no particular diagnostic point, a search must be made for the cause and the same eliminated if possible.

Gonorrhoeal Iritis is usually of the chronic type of urethritis or vesiculitis. They are slower to recover and unless the cause is removed, are sure to recur.

Tubercular Iritis is usually a nodular circumscribed iritis unless the resistance is entirely broken down and then, there is a fairly progressive loss of sight, a minus tension and, frequently, complete loss of the eye.

Sympathetic Iritis or Sympathetic Ophthalmia is an inflammation with a plastic exudate by an injury. There are several theories as to the cause, but I think the main one accepted to-day is a proteid sensitization to uveal pigment with a reaction in the sympathizing eye. The treatment for this is preventive, namely, the complete freeing of all iris adhesions in an injury or cataract extraction and early enucleation if there are signs in the other eye but, if not seen until the sympathizing eye is well established, it is better to leave both eyes in and treat the patient with massive doses of Salicylates and, recently, Doctor Verhoeff of Boston has suggested the use of large doses daily of diphtheria antitoxin to desensitize the patient. His histories have proved this to be of great advantage.

The treatment for iritis, as you can see, must be directed towards the cause but, while looking for the source of infection, a mydriatic must be used such as Atropine, Homatropine, Scopolamine or Duboisine. As a rule the patient should be kept in bed, if the infection is very acute, and in a slightly darkened room to avoid lacrimation and photophobia. He requires purgation and sometimes artificial leeching or blood letting in the neighbourhood of the temples. A few patients feel better with cold compresses and others prefer heat applied locally. Salicylates and Soda Bicarbonate usually relieve the pain no matter what the cause. While you are treating the condition, investigation should be carried along the lines mentioned above, namely, urinary examination, blood Wasserman test, genito-urinary examination, X-ray of the sinuses and teeth, washing out of the sinuses if necessary, and probing and soforth of the tonsils.

If you could see the rapidity with which

people improve when the source of infection has been eliminated, you would be most gratified.

The sequelae of iritis are very damaging to the eye. Secondary glaucoma from adhesions to the lens require an iridectomy even if the attack is still acute. Otherwise, the sight will be lost. There may be a minus tension due to prolonged inflammation without enough adhesions to produce glaucoma. The glands in the ciliary body and iris may have been so damaged as to produce a hypotony with diminished sight even without the obstruction of exudates or membrane, and a gradual loss of the eye extending sometimes over years. You can get a pupillary membrane obstructing the sight which may have to be operated on at a later date or a cataract formation due to the change in the nutrition of the lens.

Compound Fractures and Dislocations.

R. M. JANES, M.B.

THE recognition and proper care of compound injuries of bones and joints remains constantly of interest and importance. It is not only important that the surgeon should have a proper understanding of these conditions but, since the early treatment is done usually by the family doctor, the general practitioner should have a clear conception of what he would do should he be called upon to treat such a case. The ultimate result will depend upon the emergency treatment rendered and upon the length of time allowed to elapse between the injury and its adequate care.

Compound fractures are of two types, first, those produced by a penetration of the overlying soft tissues from without, and second, those produced by penetration of the soft tissues by a fragment of bone. Dislocations may be compounded in much the same way. Naturally, since the tissues are divided from without by a non-sterile object in the first type, and from within by a sterile fragment of bone in the second, the former is much more likely to become infected. Both injuries result, as a rule, in a good deal of laceration. It is important to remember that the protruding portion of bone carries infection in those fractures compounded from within.

As a rule the first treatment necessary is control of the haemorrhage. Bleeding is frequently quite free, especially if a large vessel has been torn, and even apart from this a good deal of blood escapes from the medulla. A pressure pad bandaged firmly over the wound will frequently be all that is necessary, but if this proves inadequate a tourniquet must be applied. If local pressure will suffice it is better than a tourniquet, since it does not result in a corresponding devitalization of tissue. Apart from the danger of producing a nerve injury with a tourniquet, the cutting off of the blood supply to the extremity over a period lessens the resistance of the tissues to infection. Application of a narrow tourniquet of any kind to the arm may result in nerve injury, and for this reason a broad band should be used, preferably the arm band of a blood pressure apparatus. If a tourniquet has been applied and it is necessary to transport the patient for a considerable distance, the pressure should be released for a few moments, at intervals say of half an hour.

Whether or not it is necessary for control of bleeding a sterile pad should be placed over the wound at once. If an antiseptic solution such as tr. iodine or alcohol is available it should be used. Should a fragment of bone be protruding from the wound, do not reduce it. Remember that this broken end of bone may have been driven into the dirt and that it is grossly infected. It was sterile when it penetrated the soft tissues and these are likely to be little infected. Only when the blood supply to the limb beyond the fracture is cut off by pressure on the main vessel is it justifiable to reduce such a fragment, and not then if facilities for properly attending to the fracture are within close reach. The protruding bone should be covered with a dressing and left alone.

It is wise to apply some sort of a temporary splint before attempting to move the patient. This reduces the amount of pain

that will be suffered, avoids further tissue injury, and prevents the reduction of such a fragment of bone as that mentioned above. The patient will be rendered more comfortable and the amount of shock reduced by the hypodermic administration of a quarter grain of morphine if it is at hand. This should always be done if it is necessary to transport the case a considerable distance for treatment.

The injury which has resulted in a compound fracture of one or more of the large bones may always have produced so much shock that it is necessary first to treat this, and then to treat the fracture. Should this be so the preliminary treatment outlined above should be carried out. The patient should then be put to bed and the ordinary treatment of primary shock employed, the treatment of the fracture being left until a later time. While this may be and frequently is necessary, no longer time should be allowed than is absolutely necessary to render administration of an anaesthetic safe, since the chances of healing by primary union decrease with each hour that passes.

If an X-ray is obtainable a radiograph should be taken. It is not essential, but gives the surgeon a clear idea of the condition with which he has to deal, thereby frequently reducing the time and manipulations. It also serves as a useful record of the preoperative findings.

Before an anaesthetic is administered the limb should be carefully examined. Pulsation should be felt for in the main vessels. They may be pressed upon by the bone fragments or they may have been lacerated. Sensation and muscle power should be tested in case of a possible nerve injury. If these precautions are overlooked the occurrence of gangrene of the extremity or the discovery of a nerve lesion on the day following the operation may prove embarassing.

The patient having been anaesthetized, (and it should be stated that an anaesthetic is almost invariably necessary for proper treatment of compound fractures and dislocations), the original dressings are removed by a non-sterile assistant who may also place a sterile pad over the wound and shave the surrounding skin over a wide area. All shaving and cleansing must be done away from the wound. A sterile towel or sheet should now be placed under the limb. The surgeon, who has cleansed his hands and donned a sterile gown, gloves and cap, places a fresh sterile pad over the wound and cleans the surrounding skin for a distance of ten or twelve inches on all sides of the wound with gasoline, then ether, then 2½ per cent. or 5 per cent. tincture of iodine When this is accomplished the wound is cleaned in a similar manner. It should be remembered that the mechanical cleansing is more important than the chemical and that the subsequent course of the case will depend very largely upon the thoroughness with which this part of the operation is carried out. Fresh sterile drapings should now be applied.

About one-eighth inch of the skin margin of the wound should next be removed, preferably with a sharp scalpel. Any projecting tags of tissue are cut away. If a piece of bone is projecting it is wise to curette the broken end gently, in order that any retained debris may be removed. The deeper parts of the wound are now explored; and debris is removed; tags of muscle or fascia are cut away; bleeding points are ligated. Any tissue which will not bleed when cut may as well be removed, as it is not viable, and dead tissue will furnish a culture medium for bacteria. Small fragments of bone which have no attachment to periosteum may be removed, but large pieces, and those with periosteal attachment, should be left in situ. The fracture is now reduced. It may be done easily, usually under direct vision. If tendons or nerves have been divided they should now be sutured. The wound may be closed with interrupted sutures of silk-worm gut, horse-hair or any satisfactory skin suture. It is rarely necessary or wise to suture the deeper tissues. If a considerable dead space is being left a drain may be inserted to be removed twenty-four hours later, or one end of the wound may be left open sufficiently to allow the discharges to escape. A dry sterile dressing is applied.

Any fixation may be used to retain compound fractures in position. The problem is similar to that of retaining a corresponding simple fracture except that the apparatus must be one which will allow of ready access to the wound without disturbing the position of the fractures. For the majority

of extremity fractures plaster of Paris is efficient; for fractures of the femur a Thomas bed splint is as a rule most satisfactory, and for compound fractures of the humerus a Wilson splint is very suitable. Non-absorbable materials such as bone plates, steel plates, bone or steel screws and silver wire should never be used to hold compound fractures in position, since should even a mild infection occur it will not clear up until these foreign bodies have been removed. it is rarely necessary to employ any internal fixation, but if it is found necessary the ends of the fragments may be tied together with kangaroo tendon or chromic catgut passed through drill-holes in the bones.

At the termination of the operation 1,000 to 1,500 units of tetanus antitoxin should always be administered.

The great majority of compound fractures and dislocations which come under the care of the surgeon within eight to twelve hours of injury will heal by primary union if treated in this way. They should be treated in this way up to a period of twenty-four hours following injury unless there is already obvious infection. If greater periods than this have elapsed it will probably be futile to hope to avoid infection. The fracture should be reduced and the wound left open.

On the day following operation most cases will show a slight fever, up to 100° to 101°, with a corresponding increase in pulse rate. Provided there is a progressive falling of temperature and pulse rate on succeeding days this rise is of no significance. It is due merely to absorption of protein derivatives from the wound; if, however, the next day's temperature is higher, infection probably is responsible and the wound should be examined. Mild superficial infections may occur and rapidly clear up when drainage is established.

The presence of an infection of the wound should not be considered as an excuse for delay in correction of any displacement of the bones. If a very acute infection occurs then correction of position may be delayed justifiably, but it should be remembered that bone production may be as rapid or even more rapid in the presence of mild infection and that delay in reduction will mean malunion. If the position is maintained union will usually occur. If sequestrations occur, and loose fragments are likely to sequestrate in the presence of infection, it will be necessary to do a simple sequestrectomy at a later date and the problem of correction of the position of an infected, malunited fracture will not be presented.

Notes on Some Problems in Syphilis.

E. P. SCARLETT, M.B.

THE last generation saw the discovery of the three basic factors in the clinical control of syphilis—the Spirocheta pallida and the associated dark field examination, the Wassermann test, and arsenical therapy. Superficially it would seem that we had gained the mastery of the field; but this conclusion is true only in a very limited sense. There are still many unsolved problems which must be answered before our treatment can be absolute. The recent use of malaria in the treatment of neurosyphilis has introduced biologic considerations. The three basic discoveries noted above have brought with them new problems and have compelled a revaluation of the ideas of diagnosis and treatment. Failure to deal with these new factors merely enforces the truth of Goethe's dictum that nothing is more terrifying than ignorance in action. It is proposed in this paper to discuss in summary fashion two problems which have a practical bearing on syphilis as one meets it in a clinic.

The Question of Immunity.

The first is the question of immunity in syphilis. The proper study of this subject began with the discovery of the casual organism in 1905, and the growth of the organism in pure culture by Noguchi in 1911. A vast amount of experimental work has been stimulated by the hope that syphilitic antibodies and principles of immunity with

therapeutic application would be discovered. In the problem of immunity is locked up other questions—the exact definition of the Wassermann reaction, the possibility of second infection in an individual, the proper type and disposition of treatment so that the natural defence mechanism of the individual may not be ignored, the problem of cure, the possibility of such a thing as "latent syphilis." The extensive literature on the subject of immunity is grouped into (1) the experimental work, which received its first impetus from the studies on syphilis transmission in chimpanzees by Metchnikoff and Roux about 1903, and (2) extensive clinical observations. An admirable summary of the subject by Chesney has recently appeared in monograph form (Medicine, Nov., 1926).

The result of the earlier work was to show that immunity as we know it in bacterial diseases does not exist in syphilis. Antibodies have been demonstrated in studying culture organisms, but in actual experimental work these were found to be of no significance. Zinsser showed that rabbits immunized with culture organisms developed agglutinins and lysins against the culture organisms, but not against pathogenic organisms of another strain. He found too that sera of syphilitic rabbits offered no reaction against either pathogenic or culture organisms. Kolmer and Broadwell studying the serum of syphilitic individuals at various stages of the disease could find no lytic action of the serum for organisms, and concluded the lytic type of immunity is absent in syphilis. In similar fashion all attempts to produce immunity by the injection of cultures or virus or by the passive transfer of serum from syphilitic subjects have been unsuccessful.

Not to be explained therefore by antibody formation, two distinct points of view have arisen to account for the nature of the resistance which the infected individual acquires. The first is that of Neisser, who questioned the existence of any true immunity, and on experimental evidence laid down the postulate that the acquired refractory state in syphilis means that there are persisting foci of sypilitic infection, and therefore, that reinfection assumes the complete disappearance of spirochetes; immunity disappears with the casual agent. In this he is followed by Fordyce. The other school, notably Chesney and Kemp, by their experimental work throw doubt on Neisser's view and ascribe to the infected individual a gradual increasing immunity following upon the general distribution of the syphilitic virus. Resistance is present to a high degree before the healing of the primary lesion and remains with the individual for the balance of life, though it is apparently not absolute and is subject to fluctuations. Evidence would seem to show that innoculations in later stages of the disease may produce lesions characterictic to the state of syphilis which the patient is in, but rarely to chancre and the succeeding secondary manifestations. (Finger and Landsteiner). Chesney and Kemp virtually proved their point by showing that syphilitic rabbits treated late with sulpharsphenamine were refractory to a second infection while those in which treatment is begun early were uniformly susceptible. In the case of the refractory animals no residual syphilitic infection could be demonstrated, extract of lymph nodes and all internal organs being transferred from the treated rabbits to normal rabbits without producing any evidence of infection in the latter. The refractory state, therefore, did not depend on deep-set infection in internal organs. Such observations point to a type of immunity and a natural defence mechanism in the individual.

Neisser's conclusion then is not borne out by recent experimental work, and such work also contradicts his conception of reinoculation as a test of cure in syphilis.

The exact nature of the lines of defence possessed by the animal organism is not at all clear. Brown and Pearce, studying this problem in the rabbit, point out three conditions which are evident. The first is that of latency, by which the organism may harbor the spirochetes and yet not show any manifestations of disease, indicating a defensive reaction against the growth of the spirochete on one hand, and against its toxic effects on the other. Then there is a characteristic relapsing course to the reaction, the cycle being repeated until the surviving organisms are incapable of producing further injury. Finally the way in which the disease progresses in an orderly fashion is determined

by the relative susceptibility of various tissue groups. All of which would seem to indicate that the individual can react and limit syphilitic infection, although incapable of eliminating it unaided.

This conception of latency in syphilis noted above, that is, an immunity implying a condition compatible with the life of the parasite, is spoken of by some writers as the "carrier state" (Warthin). The conception is in line with analogies in other trypanosome diseases, and in typhoid fever and malaria.

Our survey, therefore, of the present views on immunity in syphilis yields this conclusion—that the infected individual is possessed of definite lines of defence which consist probably in a gradual progressive development of an extensive tissue immunity limiting the infection. It is further possible that there is an immunization of the invading organism against the host so that a balance is struck between the two, and a "state of ideal parasitism is reached."

This conception of a definite power of immunity on the part of the individual gives rise to a question of intense practical interest in the clinical control of cases. Granting a natural defence mechanism against the spirochetal infection, how far does chemotherapy and particularly arsenic therapy interfere with or ignore such a mechanism? A large school of clinicians and experimental workers are of the opinion that pushing arsenic therapy directly antagonizes biologic immunity. Brown and Pearce maintain that if arsenic does not effect early complete sterilization, it weakens the defence mechanism by clearing up skin lesions which are the first line of resistance of the body. Other writers point out that the degree of the individual's immunity determines a cure in some cases in spite of insufficient treatment, and a malignant course in others in spite of continued intensive treatment. Capeli and Pick go further and claim that arsenic therapy failing to achieve early complete sterilization, renders the system void of its natural defence and opens the road for visceral and central nervous system involvement. Just what truth there is in such a belief is difficult to judge, but it is a stimulating point of view at least, and provokes fresh consideration of the therapeutics of the disease.

The Wassermann Reaction.

The other question, the chief lines of which can only be indicated, is the significance of the Wassermann reaction in the treatment of syphilis. A host of writers are discussing this problem, seeking to give the proper content to a positive Wassermann. The diagnostic value of the test is not so much in dispute. Although not a biologically specific test its diagnostic value and limitations are fairly well defined. The problem is rather to give the reaction a value from a therapeutic and prognostic standpoint parallel with its diagnostic significance. Three questions in particular emerge:

(1) Is the persistent negative Wassermann to be accepted as the standard of cure in syphilis?

(2) How is one to view the Wassermann-fast patient?

(3) Is the Wassermann reaction to be relied on as a guide to the amount and duration of treatment?

That the persistent negative Wassermann is not to be taken as the index of cure is the feeling of most writers. Wile says that a positive Wassermann does not necessarily mean active spirochetes any more than a positive tuberculin test means active tuberculosis in an arrested case. He feels that the majority of syphilitic cases to-day are not being treated for syphilis, but for a positive Wassermann. The proper assessment of a cure should insist that the Wassermann be taken as a symptom, that it be checked and weighed in conjunction with the history, clinical examination, and the patient's symptoms before making the final conclusion.

Most evidence would seem to be in agreement with Wile that the prognosis in Wassermann-fast cases is as good or better than in similar negative cases. The crucial question of course has not yet been answered—whether clinically negative cases of Wassermann-fast syphilis will show any higher evidence of clinical involvement at some later date than a similar group of clinically and serologically negative cases. Having satisfied the requirements—adequate treatment, clinical control of symptoms, careful clinical examination, and a long period of observation, one may then take a more confident view of the Wassermann-fast case, admitting

the possibility of spirochete carriers, or of a complete clinical cure in spite of a persistently positive Wassermann.

Most writers feel that the Wassermann reaction is not a guide to treatment, and is of no value in prognosis. The degree of inhibition of hemolysis does not parallel the clinical improvement. The presence of a positive Wassermann reaction in an otherwise healthy individual is not necessarily an indication that the patient requires further treatment. Modern practice calls for a "maximum-minimum amount" of treatment controlled by clinical examination regardless of the Wassermann reaction.

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The Chemical Control of Respiration.*

E. H. BENSLEY, 3T0 (Med.).

THE mechanism of respiration is extremely complex and involves a considerable number of muscles which act in perfect co-ordination. The respiratory act is not a fixed one, but is subject to modification; every modification involves a corresponding modification in the response of each individual respiratory muscle. This co-ordination is made possible by the presence, in the hind-brain, of a nerve centre which presides over the numerous subsidiary centres from which nerves pass to the individual muscles. The influences which determine the action of the centre are first, those of a nervous nature, namely, impulses which travel to the respiratory centre along afferent pathways, and second, those of a chemical nature, namely, the chemical conditions existing within the cells of the respiratory centre. It is the nature of this chemical control of the respiratory centre, and, through this, of respiration, which is to be discussed in this essay.

*Based chiefly on a paper by Gesell on the "Chemical Regulation of Respiration," in Physiol. Reviews, vol. V., p. 551, 1925.

There is abundant evidence of the existence of this chemical control. One of the best pieces of evidence is the well-known cross-circulation experiment in which a cross-circulation was established between two dogs in such a way that blood which had come from the lung of one dog, "A," and had passed through the heart into the cartoid artery, then proceeded to the brain of the other dog, "B." When dog "A" was placed in an atmosphere of increased carbon dioxide tension there was no change in the breathing of this animal, but the breathing of dog "B" became more marked. A change in the chemical composition of the blood supplying the respiratory centre produced an alteration in the action of the centre.

What is the nature of this chemical control? In 1905 Haldane and Priestly showed that the respiratory centre was very sensitive to changes in the carbon dioxide tension of the alveolar air and therefore of the arterial blood. In 1911 Winterstein reported the results of perfusing young rabbits with

acid solutions and concluded that it was not carbon dioxide as such, but rather the H ion concentration of the blood which serves as the chemical stimulus to respiration. Hasselbach reached the same conclusion and accordingly Haldane and his associates modified their views and gave their results in terms of arterial H ion concentration. The theory that the H ion concentration of the blood is the respiratory stimulus is still held at the present time by many investigators in spite of the fact that there is a considerable accumulation of experimental evidence which shows that there is no causal relationship between the degree of stimulation of the respiratory centre and the H ion concentration of the blood.

Scott (2), using decerebrate cat preparations, showed that the degree of hyperpnoea, on increasing the carbon dioxide content of the alveolar air, was determined by the concentration of carbon dioxide and was independent of the reaction of the blood which he varied by injecting sodium bicarbonate. Haggard and Henderson (3), (4), find hyperpnoea and alkalemia at high altitudes. Similarly the combination of hyperpnoea and increased alkalinity of the blood is found in anemia, after haemorrhage, and in fever. Evidently the relation between pulmonary ventilation and H ion concentration of the blood is as frequently an inverse as a direct one. Gesell suggests that it is the H ion concentration of the tissues of the respiratory centre which acts as a direct respiratory stimulus, and not the H ion concentration of the blood. This would not seem to overcome the difficulty since the reaction of the tissues of the respiratory centre might be expected to vary with that of the blood. However, it must be remembered that the acidity of the blood does not necessarily correspond to its acid effect on the tissues. The acidity of the blood depends on the relative proportions of acid and basic substances present in it, while the acid effect depends not only on its acidity, but also on the relative rates at which those acid and basic substances diffuse into the tissues. Thus Jacobs (5), (6), (7), has immersed tissues in solutions containing carbon dioxide and sodium bicarbonate in such proportions that the reaction was alkaline. In spite of its reaction the solution had an acid effect on the tissues as a consequence of the rapid diffusion of carbonic acid into the tissues which are readily permeable to the acid, but, on the other hand, are relatively impermeable to sodium bicarbonate. If such a solution be tasted it will be found that, in spite of its alkaline reaction, it is distinctly acid to the taste. The cells of the taste-buds are freely permeable to carbonic acid but relatively impermeable to sodium bicarbonate. The same is true of the tissues in general. With the aid of a manganese dioxide electrode placed directly in the circulating blood, Gesell and Hertzman have recorded continuously the H ion concentration of the arterial blood on smoked paper. By the use of this apparatus they found that the administration of carbon dioxide raised the H ion concentration of the arterial blood more than the venous, indicating that the tissues, including the respiratory centre, have absorbed carbon dioxide and became more acid. On the other hand injection of sodium bicarbonate produced practically the same decrease in the H ion concentration in arterial and venous blood, indicating that very little sodium bicarbonate has passed into the tissues. It has been shown by somewhat similar experiments that carbonic acid also diffuses into the living tissues more rapidly than any other acid produced in the body. From this it is evident that the acid-effect of the blood on the respiratory centre does not correspond to the acidity of the blood, but rather to its carbon dioxide content. This theory, based on the assumption that the respiratory stimulant is the H ion concentration of the respiratory centre and on the distinction between the acidity and the acid effect of the blood on the centre, explains the results of Lacquer and Verzar, who found that an increase in acidity brought about by increase in carbon dioxide concentration of the blood, produced marked hyperpnoea while injection of other acids in such quantities as to bring about an equal change in acidity, produced relatively little effect on breathing. It also explains the results of Scott (2), who found, as mentioned before, that the pulmonary ventilation depended on the carbon dioxide concentration of the blood regardless of the reaction. It does not, however, afford an explanation of hyperpnoea associated with alkalemia involving a lower-

ing of the carbon dioxide content of the blood, which occurs in anoxaemia.

Several explanations have been given of increased breathing associated with anoxaemia. Pfluger, in 1868, suggested that the respiratory centre was stimulated by circulating acid metabolites which were accumulating in the blood as a result of oxygen deficiency. This, however, cannot be the case since the blood becomes, not more acid, but more alkaline in conditions of anoxaemia. Rosenthal, in 1862, proposed that lack of oxygen stimulated the centre directly. Others have suggested that lack of oxygen renders the centre more sensitive to the H ion concentration of the blood. None of these theories have proven very satisfactory. In all of them, the metabolism of the respiratory centre itself has been ignored. The opinion held generally by most investigators is that the metabolism of nervous tissue is low. However, there is considerable evidence to the contrary. Gesell has shown that there is a close correlation between tissue metabolism and volume-flow of blood. Jensen states that the volume-flow of blood through the brain is much greater than through muscle or any other part of the body. Baglioni finds nervous tissue the seat of heat production and that this is considerably increased in activity. Moore found the rate of acid production in the intact living medulla of the frog to be about twice that of muscle. The evidence seems to indicate that there exists in resting nerve tissue a relatively high level of metabolism, and that this metabolism is increased with increased activity of the tissue. McGinty and Gesell (8), have shown that if the oxygen supply of nervous tissue is cut off, there is a rapid accumulation in the tissue of lactic acid. Normal unanaesthetized dogs were decapitated and the head split with one stroke of a guillotine. One half of the brain was promptly frozen in liquid air, the other half frozen from two seconds to thirty minutes later. They found that lactic acid accumulation was very rapid. During the first three minutes of oxygen lack the rate of formation was 20 mgms. per 100 gms. of tissue per minute. The rate then falls rapidly to zero. This experiment shows that lactic acid accumulates rapidly in nervous tissue as a consequence of oxygen deficiency brought about by completely cutting off the blood supply of the tissue in question. McGinty and Gesell also demonstrated experimentally that the lactic acid content of nervous tissue in an animal suffering from carbon monoxide anoxaemia is considerably greater than in normal animals. The results of these experiments indicate that the originators of theories explaining anoxaemial hyperpnoea were not justified in ignoring the metabolism of the respiratory centre.

On the basis of these results, Gesell has offered an explanation of the cause of anoxaemial hyperpnoea, based on the same fundamental assumption as his explanation of the relation between the carbon dioxide content of the blood and respiration, namely, that the direct regulator of the respiratory centre is the H ion concentration of the cells of the centre. Gesell points out that if the organism is suffering from anoxaemia, the tissues, including the cells of the respiratory centre, will be receiving a supply of oxygen, which is inadequate for the complete oxidation of the acid products of metabolism, of which the most important is probably lactic acid. There will, therefore, be an accumulation of these poorly diffusible acid products of metabolism in the respiratory centre accompanied by an increase in the H ion concentration of the centre. This will cause increased breathing. The greater the oxygen deficiency, the greater will be the accumulation of acid metabolites in the centre, and the more marked the hyperpnoea. This is the explanation which Gesell offers of the hyperpnoea which is characteristic of conditions of oxygen deficiency. As Haggard and Henderson (4) have pointed out, this hyperpnoea is associated with alkalemia. They believe this increased alkalinity of the blood to be a consequence of the hyperpnoea, which causes carbon dioxide to be blown off from the lungs at an unusually rapid rate, resulting in a decrease in the carbon dioxide content, and therefore in the acidity of the blood.

Summarizing these conclusions, it may be said that the direct chemical regulator of the respiratory centre is the H ion concentration existing within the cells of that centre. Alterations in the H ion concentration of the centre are due to:

(1) Changes in the carbonic acid con-

tent of the blood supplying the centre, carbonic acid diffusing more readily into and out of the centre than any other acid or any important constituent of the blood.

(2) Changes in the amount of oxygen supplied to the centre by the blood relative to its metabolic activity, since on this relation is dependent the extent of the oxidation in the centre of poorly diffusible acid metabolites into readily diffusible carbonic acid.

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Non-Infectious Leucocytosis.

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THE purpose of this paper is to point out the error of concluding that a marked leucocytosis always means an infection and to illustrate some of the more common non-infectious conditions in which a leucocytosis of the polymorphonuclear type occurs.

Definition.

The upper limit of the normal white blood count is usually considered to be 10,000 cells per cu. millimeter of blood and the highest normal differential count for polynuclears is thought to be about 75 per cent. When the lucocyte count is above 10,000 cells, a leucocytosis is said to be present.

Source of the Polynuclear Leucocyte

[1]The polymonphonuclear arises from a larger cell with similar granules; the myelocyte in the red bone marrow. After acute pneumonia of a few days' standing a considerable proportion of the yellow marrow of the femur is changed into red marrow. In acute pneumonia the number of polymorphonuclear cells in the lung exceeds by several times the total number in the blood stream.

The following is a classification of non-Infectious Leucocytosis:

1. Physiological Leucocytosis.
2. Post-hemorrhagic Leucocytosis.
3. Post-operative Leucocytosis.
4. Leucocytosis in Renal Colic.
5. Leucocytosis following the injection of certain organtic substances.
6. Leucocytosis assiciated with Neoplasm; and cachectic states.
7. A miscellaneous group:
   - I. [3]Coronary thrombosis.
   - II. Paroxysmal tachycardia.
   - III. Uraemia.
   - IV. Acute circulatory failure.
   - V. Intestinal obstruction.

The first four groups are here discussed in some detail.

Physiological Leucocytosis.

1. Infancy and Childhood.—[7]The following table shows the average white blood count and per cent. of polynuclears at this time of life:

Age	W.B.C.	% Polys
New born	20,000	75
Two weeks	10,000	50
Six months	15,000	40
One year	12,000	40
Three years	10,000	45
Six years	8,000	65

2. Latter Weeks of Pregnancy.—The average white blood count is 12,000 to 13,000.

3. Labour.—The count rises abruptly to 20,000 to 30,000, dropping to normal in a few days.

4. Digestion, Cold Baths and Strenuous Exercise.—In these conditions the count rarely exceeds 13,000.

[2]Post-Hemorrhagic Leucocytosis.

In external hemorrhage the leucocytosis is usually slight.

But, in hemorrhage into the peritoneal, pleural or joint cavities or about the dura mater the count may go up to 20,000 or 30,000 cells with a polymorphounclear differential count of 80 per cent. to 90 per cent.

The peak is reached about ten hours after the hemorrage and persists for four to five days.

In a series of twenty-eight cases of ruptured ectopic gestation the average white count during the first day was 20,000.

[4]The following is a report of the leucocyte count taken on 150 patients before they were operated on for ectopic gestation. These patients were divided into three different groups as follows:

Group (a). Those with a normal leucocyle count.—Seventy-two patients fell into this group. But, in all this series it was found at operation that either no rupture had occurred or else it had been walled in, that is to say no fresh blood was in the pelvis in any case.

Group (b). Cases with a leucocyte count of 10,000 to 16,000.—Fifty-five cases in this group. Only nine per cent. of these cases were unruptured and showed no fresh blood in the pelvis.

Group (c). Patients with a count of 16,000 or over.—Twenty-three cases in this group. At operation it was found that a rupture or incomplete tubal abortion with active bleeding into the pelvis had occurred in all these patients. The differential count ranged from 77 per cent. to 96 per cent. for polynuclears in this group.

According to [3]Pepper every case of intradural hemorrhage shows a marked polymorphornuclear leucocytosis within a few hours.

I would like to suggest here that a leucocyte count should be valuable as an aid in the diagnosis of a ruptured spleen or liver if the case did not come under observation till a few hours after the accident.

Post-Operative Leucocytosis.

[5]Witter studied the leucocyte count in a series of thirty-nine cases having 'clean" operations i.e. cases in which no infection was known to be present before the operation and in which no complicating events followed the operation.

In twenty-five of these cases an abdominal hysterectomy was performed and in the remaining cases a dilatation and curettage of the uterus was the operation.

The following is a table of the leucocyte count found before the operation and each day for the first seven days following the operation:

Day of Count	Cases	Min.	Max.	Average
Pre-Operative	30	6,000	13,000	9,000
Post-Oper. 1st	21	12,000	26,000	18,000
" 2nd	24	10,000	27,000	16,000
" 3rd	28	9,000	21,000	13,000
" 4th	25	6,000	21,000	11,000
" 5th	23	7,000	19,000	10,000
" 6th	23	8,000	19,000	12,000
" 7th	18	7,000	24,000	13,000

The average per cent. of polynuclears in the pre-operative count was 66 per cent. and on the first day post-operative was 82 per cent. The per cent. gradually fell to normal.

Cabot found the count in a patient after ether anaesthesia to be 7,200. But, after an operation the count rose to 27,300.

The leucocytosis is believed to reach its height about the fourth hour after operation.

The following is the count which I made on a patient who had a dilatation and curettage performed and a small cyst removed from the ovary: The pre-operative count was 4,400 and eight hours after the operation the count was 14,400 with a differential count of 89 per cent. polynuclears.

Leucocytosis in Renal Colic.

I examined the case records of all the public patients admitted to the Toronto General Hospital for the last year with a diagnosis of renal colic. There were twelve cases. In six of these cases there is no record on the chart of a white blood count. The following is a brief account of the remaining six cases. I go into some slight detail in recording these cases so that one can judge whether or not an infection is also present e.g. it is necessary to show that a pyonephrosis is not present:

Case 1:—Male; age 36.

Complaint:

1. Severe pain in lower left quadrant of abdomen extending through to back. Duration 6 hours. Patient was perfectly well till he was suddenly seized with pain.

2. Vomiting 5-6 times in last three hours.

The whole left side of abdomen and back was tender especially in costo-vertebral angle.

The temperature varied from normal to 99.2°F.

The pulse varied from 60 to 80.

The urine contained a trace of albumin and 5 to 8 R.B.C. per H.P. field. Otherwise it was normal.

The white blood count was 15,200 and the differential count showed 94 per cent. polynuclears.

The pain suddenly disappeared nine hours later.

Case 2:—

Just the following facts were obtainable in this case:

Tem. 99.5; pulse 80.

Urine contained albumin + and W.B.C. ++, otherwise normal.

The white blood count was 23,000 on the day after admission and two days later it was 19,000.

Case 3:—Male; age 23.

Complaint:

1. Severe continuous pain in lower right quadrant of abdomen extending around to the back. Duration six hours. Pain began as a dull ache.

2. Vomited twice.

Never ill before. Physical exam. negative except for severe tenderness 3" to right of umbilicus and behind at the same level. No urinary symptoms.

Atropine grs. 1/100 gave relief from pain.

Temp. 99; pulse 70.

Urinalysis showed a trace of albumin and 1 to 5 R.B.C. per H.P. field. Otherwise normal.

The white blood count was 16,000. There were 82 per cent of polynuclears.

Three days later patient discharged free of pain.

Case 4:—

Complaint: Severe pain in right side. Duration four days.

Urinalysis: Many pus cells and occasional R.B.C.

The white blood count was taken two days later and was 9,100. Polynuclears 75 per cent.

The stone was subsequently removed from the ureter.

Case 5:—

Complaint:

1. Colicy pain in right lower abdomen. Duration one day.

2. Unable to void for thirty hours.

3. Nauseated.

Similar attack for two days one year ago.

Physical examination negative except for voluntary rigidity and slight tenderness in right lower quadrant of adbomen.

Patient catheterized and the specimen was normal.

A cystoscopic was done and an obstruction was met by the right ureteral catheter but the operator was able to get the catheter past the obstruction and urine was obtained from the right pelvis. It contained many red blood cells and crystals but no pus cells.

The white blood count was 20,000.

Case 6:—Male; age 28.

Complaint:

1. Sudden pain in right side of abdomen. Duration seven hours.

2. Urinary frequency.

3. Vomiting.

Physical Examination: Tenderness in lumbar region and costo-vertebral angle. Otherwise negative.

Pulse 105; temp. 99.1.

Urine contained some red blood cells and two white cells per H.P. field.

The white blood count was 15,000.

The X-ray diagnosis was a ureteral calculus on the right side.

My conclusion is that a polymorphonuclear leucocytosis varying from 15,000 to 20,000 is the rule in renal colic.

Leucocytosis Following the Injection of Certain Organic Substances.

The leucocytosis is usually slight.

Some of the substances which will cause it are collargol, turpentine, camphor, antipyrin, digitalis and arsphenamine.

Neoplasms and Cachectic States.

The leucocytosis is usually moderate and is more apt to be present if the mass is ulcerating.

The leucocyte count is an aid in the differential diagnosis between Addison's Pernicious anemia and carcinoma of the stomach. In the former there is a leucopenia or a normal count.

Miscellaneous Groups.

In coronary thrombosis the leucocytosis may be marked and care must be taken lest this disease be mistaken for an acute abdominal condition.

In paroxysmal tachycardia the count may exceed 20,000.

In some cases of intestinal obstruction[6] the count is said to rise to 16,000 to 20,000.

My conclusion is that there are certain common non-infectious diseases in which a marked leucocytosis of the polymorphonuclear variety occurs and that these conditions should be borne in mind in the interpretation of such a leucocytosis.

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Retention of Urine—Its Causes and Management.

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The causes of retention of urine may be classified according to the following table:

I. CONGENITAL.

1. Phimosis.
2. Atresia of meatus—Imperforate meatus.
3. Valves in posterior urethra.

II. ACQUIRED.

1. **Inflammatory.**
   - (a) Acute.
     - i. Prostatic abscess.
     - ii. Cowper's gland abscess.
     - iii. Ischio-rectal abscess.
   - (b) Chronic-Stricture.
2. **Tuberculosis**—Stricture of urethra.
3. **Syphilis**—Spinal Cord Disease.
4. **New Growth.**
   - (a) Prostate.
     - i. Adenomyoma.
     - ii. Carcinoma.
   - (b) Vesical tumour.
5. **Calculus.**
   - (a) Vesical.
   - (b) Urethral.
6. **Trauma.**
   - (a) Ruptured urethra and stricture.
   - (b) Spinal cord injury.
7. **Diverticulum** of the bladder.
8. **Pressure** from tumour or mass outside the urinary tract.
9. **Post-operative.**
10. **Hysteria.**

(Of this group the most common causes are the **inflammations** or **abscesses** occuring mostly **before** fifty years of age and **stricture** and **enlarged prostate** occurring after fifty years of age).

I. **CONGENITAL**—Phimosis and atresia of the meatus are rather uncommon, but are conditions which one must be on the lookout for. Congenital valves of the posterior urethra are also very uncommon, characterized by a large lower abdomen and incontinence of urine.

II. **ACQUIRED.**

1. **Inflammatory; (a) Acute; i. Prostatic Abscess.**

A prostatic abscess usually has a history of an acute urethritis of from ten days to three weeks' duration. The patient notices that the discharge has stopped rather suddenly and is greatly pleased. On questioning, one finds that with the stoppage of the discharge, the patient has been having to urinate a little oftener and there is a slight straining in order to void. Rectal examination will show the prostate to be enlarged in one or both lobes, smooth, firm, very hard, and excruciatingly tender. Hot Sitz baths may be commenced at once and the acute prostatitis arrested, or it may progress, causing more and more difficulty until the patient is unable to urinate.

Staphlococcus infections set up a focus in the prostate secondary to a pimple or boil, infected finger, etc., which manifests itself by difficulty and retention. The diagnosis is made by rectal examination.

The treatment of the retention is catheter. It is contraindicated to pass any instrument in the presence of an acute urethritis. This is the one exception when the practitioner is forced to pass a catheter through the acutely inflammed urethra. The abscess must be drained. This can be done by a perineal incision or rupturing through the prostatic urethra with a sound, using the finger in the rectum as a guide for the sound in the urethra. The perineal incision is the better method.

ii. **Cowper's Gland Abscess.**—This also occurs secondary to an acute urethritis and manifests itself by pain and tenderness in the perineum, anterior and lateral to the rectum. An area of induration and slight redness is found. Rectal examination is very tender. The two areas of Cowper's glands are palpated with the index finger in the rectum and the thumb outside and any induration and tenderness noted. The treatment of this condition is incision and drainage.

iii. **Ischio-rectal Abscess.**—Occasionally one finds an ischio-rectal abscess which burrows around anterior to the rectum and causes sufficient pressure on the membranous urethra to give difficulty of urination and retention.

(b) **Chronic Inflammation—Stricture.**—This is one of the real common causes of retention of urine. It is evidenced by a decreasing size and force of the stream with an increasing difficulty and frequency. The "gleet" so commonly present is usually an evidence of a stricture. The difficulty increases accompanied by great frequency until the urine is forced out drop by drop after a great deal of straining. The urine voided into two or three glasses is clear with shreds in the first which contain pus and secondarily infecting organisms. Later, when cystitis develops, both specimens become cloudy with pus and shreds.

The diagnosis of stricture is made by the passage of a bougie, a bule, a catheter or a sound and rectal examination. Any instrument passed into the urethra in the presence of retention from a stricture will go as far as the obstruction. A good deal of care must be exercised in not forcing the sound in an effort to pass the obstruction. Rectal examination shows a chronic inflammatory prostate which is small, indurated, hard, but the hardness is not that stony hardness of a carcinoma. Above the prostate one can feel the distended bladder which must not be confused with an enlargement of the prostate.

What is to be done to relieve the retention due to stricture? Drainage must be established and this is done best by the use of filiforms and Le Fort followers. Several filiforms are selected and passed into the urethra. First one is manipulated into every possible corner of the stricture in an effort to pass it. Sometimes there are small pockets in the urethra distal to a stricture and the end of the filiform engages in it. The first filiform is left in position and another introduced. This is manipulated into every possible position in an effort to get it beyond the stricture. More filiforms are added until one has four or five in the urethra with each tip against the stricture. Taking each filiform in turn it is pushed in in an effort to find the stricture. If this is unsuccessful, the filiforms are withdrawn and some paraffin oil introduced into the urethra by a bulb syringe, using sufficient force to send the oil through the stricture and lubricate it. The filiforms are introduced again and the procedure repeated. By patiently trying one

filiform after another, one will usually pass. A Le Fort follower (or, as they are usually called, a "follower"), is then screwed into the end of the filiform. The follower is guided to the proper opening of the stricture by the filiform and on through into the bladder by the same manoeuvre as one uses in passing a sound. The filiform curls up in the bladder.

How many followers and what sizes should be passed? At the first sitting, particularly if an anaesthetic is not used, one No. 16 F. follower is sufficient. One is concerned in producing sufficient dilatation to enable the patient to urinate. As little instrumentation as will establish urination should be carried out at the first sitting. Prolonged instrumentation and rapid dilatation of a stricture in the presence of an infected urine tend towards production of a chill. If the patient is under an anaesthetic one is justified in dilating the urethra to the full size unless the condition of the patient is poor and the urine is very badly infected.

Dilatation of the stricture is carried out at intervals of three or four days until the normal size of the urethra is reached; usually taken as No. 26 F. One may use filiforms and followers or sounds for this dilatation, but it is safer to bring the dilatation up to No. 26 F. with the filiforms and followers and then the later instrumentation can be carried out with sounds. A good rule for further dilatation is, once a week for a month, once a month for a year, and once a year for the rest of the patient's life. This rule is elastic according to the results obtained, but the importance of dilatation over a period of years should be stressed.

2. **Tuberculosis.**—Young men under twenty years of age with renal tuberculosis have a tendency to the formation of a urethral stricture. These are uncommon and must be treated by dilatation, first with filiforms and followers, and later with sounds.

3. **Syphilis.**—Spinal cord diseases of which Tabes dorsalis is a good example, produce what is commonly called a "cord bladder," that is, lack of bladder control due to disease or injury of the spinal cord. Diagnosis is made by the history and examination. Rectal examination of a patient of this type will show great relaxation of the tone of the rectal sphincter. Hence, as a routine, it is very important indeed to note the tone of the sphincter when one is making a rectal examination.

4. **New Growth.**—The type of new growths which cause retention of urine are those in the prostate and bladder.

(a) **Prostate; i. Adenomyoma.**—Benign hypertrophy or adenomyoma of the prostate occurs in men over fifty years of age. There is a gradual onset of delay in starting the stream and he has to strain for several seconds before the stream reaches the end of the penis and drops down perpendicularly with no force to project it. The residual urine usually becomes infected in the bladder and introduces the symptoms of frequency and occasionally pain during and toward the end of micturition. Haematuria occurs also and relatively more often than it does with carcinoma of the prostate. Finally, the patient is exposed to an extra amount of cold, has a chill and retention supervenes. Also, overdistention of the bladder owing to neglect to empty the bladder at the proper time or an indiscretion such as an alcoholic bout, may be followed by complete retention in these patients.

The diagnosis is made by examination of the prostate. Rectal examination is carried out in the routine way, the anus inspected, the tone of the sphincter noted, the membranous urethra, area of Cowper's gland, prostate and rectum palpated as far as the index finger can reach. The adenomyoma in the prostate has the medium furrow present, the lateral lobes are enlarged, smooth, firm on pressure and elastic. If there should be any area of induration it must be examined very carefully, as a stony, hard area indicates a carcinoma. Sometimes very slight enlargement of the prostate produces retention as well as the greater enlargements. The difficulty and later retention is due to a lateral pressure on the prostatic urethra by the median lobe. This is sessile and has too thick a base to move over the internal vesical orifice and act as a ball valve. Very rarely do we see a median lobe with a pedicle.

Prostate; ii. Carcinoma.—Malignant enlargement in the prostate has the same symptoms as the benign growth, but has its aver-

age age as sixty-five. Haematuria occurs less frequently with this type than with the adenomyoma. The diagnosis is made by rectal examination. Usually it is difficult to outline the prostate, median furrow is gone, the surface is rough and the rough areas stony hard. When the index finger presses one of the areas of roughness it gives the impression of feeling a very hard, board-like surface. As the growth progresses it infiltrates laterally, upwards and downwards to fix the prostate in position, and infiltrates the area of the seminal vesicles and base of the bladder. In a few cases, there is an infiltration down over the membranous urethra. Also, one occasionally sees a carcinoma of the prostate which is smooth and hard without being rough.

The treatment of the retention due to enlarged prostate is the removal of the urine by whatever way possible. The simplest method should be tried first and that is hot bath. Failing this, the catheter should be used.

One must keep several different types of catheters ready and sterilized at the time of catheterization so that each instrument in turn can be tried quickly. In this way, the different catheters may be passed without undue delay and exposure of the patient. in order, one should try, (1) a soft rubber catheter, (2) Coude or gum elastic catheter, (3) catheter on a stilette, (4) metal catheter with a postatic curve. Failing introduction of a catheter suprapubic puncture may be done as a temporary method until suprapubic cystotomy may be carried out. The catheters used should be 18, 20, or 22 French in size. Suprapubic puncture is done at a point half an inch above the symphysis pubis and the trocar and cannula pointed backward and slightly toward the perineum. A lumbar puncture needle may be used for suprapubic puncture and has the advantage of being smaller than most trocars.

Morphia should not be used for retention of urine unless one has failed in the method cited above and it is necessary to wait until operative proceedures can be commenced.

(b) **Vesical Tumour.**—Bladder tumours more often produce a clot retention during the time when the tumour is bleeding profusely. Occasionally a tumour is found on a pedicle which will float across the internal vesical orifice during the act of micturition and close the orifice. Temporary treatment is catheterization. The tumour must be attacked and removed. Most benign tumours can be treated through the cystoscope and the larger malignant ones treated by resection, diathermy or radium through a suprapubic cystotomy wound.

5. **Calculus.**

(a) **Vesical Calculus.**—A stone in the bladder may be small or large and produce the usual symptoms of cystitis. A stone small enough to be passed through the urethra may give a little vesical irritation evidenced by frequency, discomfort in the perineum referred along the uretha to the tip of the penis. The larger stone is accompanied by a good deal of frequency and pain referred to the end of the penis. A partial retention may occur with a stone in the bladder when it floats over the internal vesical orifice during urination and causes the stream to cease suddenly. The patient alters his position by lying on the side or face, the stone floats away from the orifice and the bladder empties.

(b) **Urethral Calculus.**—The stone is floated into the urethra by the stream of urine and is passed or obstructed there. When a small stone is passed through the urethra, there is a sudden hesitancy during the flow of the stream which is suddenly released as the calculus comes from the meatus. A small quantity of blood follows the calculus.

The diagnosis of the stone in the bladder is made by the use of the cystoscope or x-ray. If the x-ray is used, one must be very careful that the shadow found is within the bladder and not in the rectum. A stone impacted in the urethra must be diagnosed by palpation, bearing in mind that the urethra can be felt in its whole length except the first inch from the bladder outward.

The immediate treatment of retention due to vesical calculus is catheterization. A type of catheter such as a Coude may be stiff enough to push the stone away from the orifice and empty the bladder. Later the stone must be removed either by lithopaxy or suprapubic lithotomy. A small stone impacted in the urethra may be removed by the urethroscope, external urethrotomy

or pushed back into the bladder and treated as a vesical calculus.

6. **Trauma.**

(a) **Stricture and Ruptured Urethra.**—The diagnosis and treatment of traumatic stricture is the same as that of inflammatory stricture.

Ruptured urethra is caused by falling astride a fence, a bar or some hard object. Fractured pelvis is frequently accompanied by ruptured urethra. Blood appears in the meatus as the first symptom. When the desire to void comes, the urine comes with difficulty or the patient is unable to start the flow. When an attempt to urinate has been made some urine and blood is forced out into the perineum under Colles' fascia and slightly distends it. This shows a swelling limited by the attachment of the fascia to the posterior edge of the trigone and the ascending rami of the ischium. Each time an attempt is made to void more urine is forced under Colles' fascia and extravasation is produced under the continuation forward of the fascia around the scrotum, penis and later up the abdominal wall.

How may the retention be relieved? Catheterization must be attempted and the greatest care exercised. The list of catheters must be used and an effort made to get one into the bladder. The most useful instruments are the catheter on a stylette or the metal catheter, as the point of the catheter can be guided by the hand to different points to find the opening leading to the bladder. The finger in the rectum may be of aid as a guide for the catheter attempting to pass an obstruction in the membranous urethra. If one is fortunate enough to get a catheter into the bladder it must be fastened in with adhesive attached to the penis. The proper point for a retention catheter is determined by withdrawing the catheter slightly until the urine stops flowing and pushing it in an inch. This places the eye of the catheter one inch inside the bladder sphincter. When the catheter has been fastened in position an irrigation is used to see if the catheter works well in the position selected.

Having placed the catheter in position an incision is made in the mid-line of the perineum "down to" the area of rupture. This incision allows the blood-clot and urine under Colles' fascia to escape to the surface. This is a very important step and one will not make a mistake by carrying it out though occasionally it may seem unnecessary especially if the catheter has been inserted very soon after the accident.

If one fails to put in a catheter one must do an external urethrotomy, opening down to the area of rupture, and pass a catheter through to the bladder. The urethra is cut completely across occasionally and it is extremely difficult to find the proximal part of the urethra. Much time may be saved if "retrograde catheterization" is done after a short search. This means passing a catheter down the urethra through a suprapubic opening in the bladder to show the position of the proximal end of the urethra and a catheter through the meatus to the distal cut end. They can be sewn together and the catheter from the bladder passed through the penis to the meatus and fastened in position. The urethral mucosa grows very rapidly when it is injured and will regenerate its tube provided a bridge of tissue is left across the area of rupture.

(b) **Spinal Cord Injury.**—Injury to the spinal cord often includes an injury to the nerves to the bladder and retention has to be relieved. Usually there is no difficulty in catheterization. Permanent drainage through a catheter or interval catheterization must be used. Automatic emptying of the bladder also occurs.

8.. **Diverticulum of the Bladder.**—A diverticulum of the bladder may be so situated that its sac burrows down behind the bladder and in frond of the rectum to reach the region of the bladder neck. In this position, the distended diverticulum exerts a pressure on the vesical orifice in such a way as to cause retention. This condition is very uncommon. The treatment is catherization and later the diverticulum must be removed.

9. **Pressure From a Tumour or Mass Outside the Urinary Tract.**—Tumour masses may be so situated as to produce pressure on the bladder neck and cause retention. This is true of tumours behind the rectum. They bulge forward and press on the rectum and, if large enough, on the neck of the bladder. Faeces collect above the point of constriction of the rectum and add their pressure to that

of the tumour to assist in producing retention. The immediate treatment is catheterization.

10. Post-Operative.—Retention of urine following operations on the pelvic viscera is a frequent occurrence. This is due to a temporary derangement of the reflexes from the pelvic viscera. It usually adjusts itself in a few days but must be treated temporarily by catheterization.

Overdistention should be avoided in post-operative retention. The injury caused to the bladder mucosa is sufficient to give a very severe cystitis. The cystitis which follows post-operative retention is rarely due to infection carried into the bladder by the catheter but the organisms normally excreted by the kidney gain a foothold on the injured bladder mucosa. Thus, we have a descending infection which becomes implanted in the injured bladder mucosa.

11. Hysteria.—It occasionally happens that an individual who lacks good nervous control may allow the bladder to become overdistended due to the inconvenience of voiding. When an effort is made to initiate the act of micturition the pain may be so severe that the patient prefers the pain of the overdistention to that of attempting to void. Urine continues to increase the distention and an increasing effort is required to void. A viscious circle is produced until the bladder is so over-distended that it can no longer contract and must be emptied by means of a catheter. A man may have retention in a similar way following an alcoholic bout.

Conclusion.

(1) A routine rectal examination should be made on all cases of retention.

(2) Men under fifty years of age have inflamation as the common cause of retention.

(3) Men over fifty years of age have stricture and prostatic enlargements as the most common causes of retention.

Medical Society Meeting—Speaker: Dr. Schick

THE following is a brief outline of the delightful address which Dr. Schick gave before the Medical Society on Thursday, February 17th, 1927, at 4.15 p.m., in Room 19, Medical Building.

In considering the influences which caused him to take up the study of medicine the speaker felt that the chief factor was his admiration for a near relative of his who practised in his home town. This man was a well-beloved general practitioner of the old school who made every effort to keep up with the times by an annual visit to the neighbouring universities. He loved his work and showed no taint of jealousy for those who were engaged in scientific medical research.

Dr. Schick's definition of a good teacher is one who makes the student feel that his own subject is the best field in medicine and consequently stimulates the student to study that particular field. Each branch of the profession is equally important, whether it be the general practitioner in personal contact with the public or the research worker alone in his laboratory. These and others are all essential units in the great medical service of to-day.

The average medical student starts his course feeling that he knows very little. His opinion of his own ability and knowledge rapidly mounts till it reaches a climax when he gets his diploma. It receives a severe jolt when he tries to put his knowledge into practice, but gradually self-confidence derived from experience returns till a second climax, not so high as the first, is reached. From here the medical man gradually becomes more modest throughout life as his experience broadens and his respect for Mother Nature increases. The older practitioners of to-day have seen great changes in the teaching of Medicine in the last score or more of years. "The old order changeth, yielding place to new." This doubtless adds to their humility.

It is interesting to note the difference between the text-book type of disease and the disease as found in practice. Not long ago a doctor writing in a humorous medical

publication of Munich, stated that he had seen a typical text-book case of Lobar Pneumonia; the first case that he had ever seen.

In practice Medicine is still an art; although it is becoming more and more scientific. A mathematician would laugh at the idea that it is a science. This is emphasized by the fact that a man with not much scientific knowledge but with a good personality can often succeed much better than one with much scientific knowledge and an unattractive personality. It is hard to teach Medical Science, but it is harder to put it into practice.

The future of the medical profession is roughly divided as follows:

(1) The treatment of individual ill patients by the family physician. This type is, sad to say, tending to die out.

(2) Specialties.

(3) Pure research.

(4) Public Health and Preventive Medicine.

The disadvantage in this last division is that it tends to State Medicine and largely destroys the personal contact between patient and physician. Nevertheless we must remember that it is easier to prevent disease than to cure it. The physician cannot cure disease; he can only aid nature in making her own cure; in other words, the physician is not "magister naturae"; he is only "minister naturae."

Dr. Schick spent a very busy time in Toronto. Besides attending Dr. Parsons' chest clinic at the H.S.C. and visiting the wards of the H.S.C., he addressed an audience on Wednesday evening, Feb. 16th, on "Icterus Neonatorum," and on Thursday evening, Feb. 17th, he addressed the section of paediatrics of the Academy of Medicine on "Tuberculosis in Childhood."

TO A YOUNG PHYSICIAN.*

The paths of pain are thine; go forth
With healing and with hope;
The suffering of a sin-sick earth
Shall give thee ample scope.

Smite down the dragons fell and strong,
Whose breath is fever fire;
No knight of table or of song
Encountered foes more dire.

The holiest task by heaven decreed,
An errand all divine,
The burden of our mortal need
To render help is thine.

No crusade thine for cross or grave,
But for the living man;
Go forth to succour and to save
All that thy skilled hands can.

Before the unveiled mysteries
Of life and death go stand,
With guarded lips and reverent eyes
And pure of heart and hand.

So shalt thou be with power endued
Of Him who went about
The Syrian hill-paths doing good
And casting devils out.

The Holy Helper liveth yet,
Thy friend and guide to be;
The Healer by Gennesaret,
Shall walk the wards with thee!

John G. Whittier.

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*From Dr. James S. Sprague's "Medical Ethics and Cognate Subjects," published by Charles P. Sparling, Toronto, 1902. Read to the graduating year by Mr. I. H. Cameron on the occasion of his last lecture, March 1, 1927.

BIGGER AND BETTER THAN EVER

THE WINNING SKIT, "A Student's Dream of Avarice." Left to right: D. S. Hoare, the night-watchman; A. R. J. Heffering and N. M. Wrong, stage hands who butted into the picture; M. M. R. Hall, one of George's gang; W. H. Masters, George, the chief rum-runner; T. H. Belt, the stude himself; C. A. Brownson, pianist; A. J. Fisher, a rum-runner; R. H. Meredith, one of George's gang; E. A. Moore, one of George's gang; G. C. Large, soloist; absent: O. A. Kilpatrick, Deputy Inspector of Custime, and H. E. Rykert, Inspector of Customs.

THE MEDETTES PRESENT "IODINE" (A TALE OF IRISH LUCK).
TOP ROW, left to right—M. H. Grant, village fiddler; M. V. Bates, G. E. Mahon, A. M. D. Knox, C. A. Fraser, M. D. Laird. (Mickey the Village Lout); R. K. Haight (Iodine, the Village Queen); M. Waldman, M. R. Cairo, B. Wilensky, M. F. Batt. LOWER ROW—K. W. Baldwin, a Ragged Urchin; G. E. Mulock, (Patrick Dooley); M. B. Brown, Sergeant of Irish Police; E. A. Gee (Molly O'Grady, a Widow).

DAFFYDIL, FEB. 10th AND 11th, 1927

T. H. BELT (TOM).

Chairman of Daffydil Committee, 1927; President-elect of Medical Society. Good luck next year, Tom.

O. A. KILPATRICK (KIL).

"The best actor in Medicine" and "the moving spirit of Daffydil." Author of two winning skits.

F. H. VAN NOSTRAND as Professor Graham. He is Quizzing the Boys.

L. A. CLARKE as Professor Rudolph. He was guiding a ship past the reefs and lost his moustache. This one is a substitute.

General Skit

See next page

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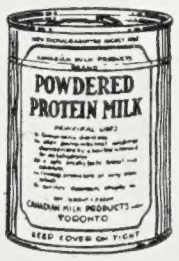

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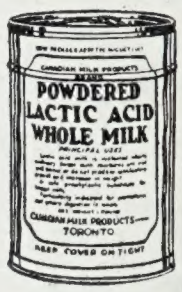

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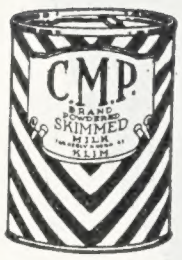

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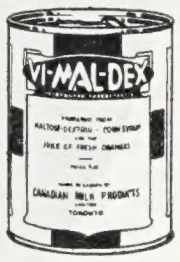

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GENERAL SKIT, "Reel Stuff." Left to right—L. A. Clarke as Professor Rudolph (without moustache); F. H. Van Nostrand as Professor Graham; Stan. Teskey as Dr. Wm. Magner; A. L. Morgan, author of Skit and moving picture expert; G. H. Jackson as Dr. Alan Brown. (He has just called for a stool). M. D. Epstein, the Sheik, Ajhab Kha; W. R. Aberhart, Miss Steppe, movie star; E. B. Patterson as Dr. F. F. Tisdall; J. E. Matheson as Dr. Jno. Oille; J. R. Westheimer, ukulele expert; absent: A. A. Overholt, Director of Movies, and O. A. Kilpatrick as Dr. Harold Ball.